THE PATHOLOGY AND PREVENTION OF VOLKMANN'S ISCHAEMIC CONTRACTURE

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Ischaemia is a rare complication of injury to a limb but must be excluded in every case. Two distinct types occur: Type I, where a proximal arterial injury gives rise to ischaemia distally; and Type II, where a direct injury gives rise to ischaemia at the site of the injury. Whatever the nature of the insult, an ischaemic contracture only develops as a result of swelling of the soft tissues where these soft tissues are contained in an unyielding osteofascial compartment. This secondary ischaemia can only be relieved by a timely fasciotomy. The diagnosis of ischaemia in an injured limb and the indications to operate on it can usually be made on clinical grounds alone.

A hundred years ago Volkmann described a contracture which involved the muscles and nerves of an injured limb. He thought the contracture was ischaemic in origin and was caused by the pressure of an unduly tight bandage. This concept of external pressure became generally accepted, but during the subsequent fifty years opinions changed.

Attention was drawn (Littlewood 1900) to the soft-tissue “effusion” that occurred in an injured limb and it was thought that it was the pressure of this swelling beneath the deep fascia that occluded the circulation. Murphy (1914) thought that this pressure obstructed the venous circulation and advocated splitting the deep fascia to relieve it.

During the First World War the problems of arterial injuries focused on the phenomenon of arterial spasm. Experience showed that the outcome was uncertain: the limb might develop gangrene or might recover completely. The mechanism of development of the arterial spasm was ill-understood but, largely due to the work of Leriche (1928), it became accepted that it was caused by a nervous reflex mediated through the sympathetic nervous system occluding both the main vessel and the collateral branches. Treatment was therefore directed towards interruption of this reflex arc by sympathetic block or arterial stripping. These procedures became widely practised in the management of arterial spasm.

In 1940 Lloyd Griffiths asserted that Volkmann’s ischaemic contracture was due solely to an arterial injury with reflex spasm of the collateral vessels. He dismissed all consideration of venous occlusion or subfascial haematoma and he thought that tight splintage was “merely contributory”. His conclusions were generally accepted and repeated in almost every paper on this subject over the subsequent twenty-five years.

It was after the Second World War that the work of Kinmonth, Simeone and Perlow (1949) demonstrated that it was impossible to precipitate arterial spasm by sympathetic stimulation, either directly or reflexly, and that it was impossible to relieve it by sympathetic interruption, yet the practice still persists today; this futile time-wasting manoeuvre should finally be abandoned.

It was the Korean War that crystallised our ideas on closed arterial injuries. We learned that a direct blow or a traction injury to an artery might cause an incomplete tear of the vessel wall. An intimal tear allowed stripping of the intima, shearing off the entrance to collateral branches; the exposed media provided a raw surface on which a thrombus would form, and as the thrombosis extended it blocked not only the main vessel but the collateral branches as well. We were taught to explore such vessels, to excise the damaged segment and, after extracting the thrombus, to restore continuity. This has now become an accepted part of acute vascular surgery, but the expertise of vascular surgeons is to no avail unless orthopaedic surgeons, who see these injuries because of the more obvious bony injury, can learn to make the diagnosis of arterial damage.

There must be a degree of traction injury to an artery that does not tear the vessel but simply stretches it. To understand the effect of such a stimulus we must consider the physiology of the normal arterial wall (Fig. 1). Normally an arterial wall is in a state of tension \( T \)

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T = P \cdot R
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where \( T \) is the active tension of the muscle coat and the stretching of the elastic coat by the mean blood pressure \( P \) in a vessel of radius \( R \).

Fig. 1

Based on a Hunterian Lecture to The Royal College of Surgeons of England April 12, 1978.
which arises in two main ways: the active tension of the muscle coat exerting the normal vasomotor tone and the elastic tension due to the elastic coat being stretched by the mean arterial blood pressure \(P\). Such an arrangement allows physiological variations of blood pressure to be accommodated by variation in the tension of the vessel wall and variation in the radius of the vessel \(R\). These functions are simply related by Laplace’s formula \(T = P \cdot R\). If any of these factors falls to an unphysiological level the system becomes unstable and the vessel collapses, a phenomenon known as critical closure (Burton 1951). Blood flow will cease before the blood pressure has fallen to zero.

A traction stimulus applied to an artery, either at the time the bone is broken or when the fracture is manipulated, may stretch the muscle coat and stimulate it to contract. The muscle fibres are capable of shortening to one sixth of their original length. If the calibre of the vessel narrows to this degree, \(R\) will fall to an unphysiological level, the vessel will collapse and blood flow will cease. Such critical closure can only be diagnosed by exploring the vessel and increasing its diameter again by injecting a bolus of fluid through the narrow segment (Mustard and Bull 1962). This restores \(R\) to Laplace’s equation. It seems logical to reverse a purely physical deficit by purely physical means and, in my experience, this technique is more likely to be successful than the traditional application of papavarine to the vessel wall. If the technique fails to dilate the vessel, one is not dealing simply with critical closure but with the collapse of a vessel distal to an intimal tear. This will therefore direct attention to the lesion which needs repair.

**THE DEVELOPMENT OF AN ISCHAEMIC CONTRACTURE**

A severe ischaemic insult may have three possible outcomes. There may be complete recovery, or gangrene may result, or it may pursue a middle course and a contracture may develop. The difference between gangrene and contracture is not a purely quantitative one. The two conditions are significantly different; gangrene involves all tissues, particularly the most distal—the fingers or the toes—and rises to a level determined by the level of the arterial insult. A contracture is a “selective” ischaemia (Eastcott 1973) of the muscles and nerves of the distal segment of a limb—the arm below the elbow or the leg below the knee. The most distal tissues—the hand or the foot—are not usually ischaemic. They are numb and paralysed, but this is due to ischaemia of the muscles and nerves more proximally. It is significant that when a contracture follows the use of unduly tight splints (Aggarwal, Singh and Gureja 1969) the ischaemia always develops in the distal segment of the limb regardless of whether the splint has been applied above or below the elbow or the knee. Seddon (1956) showed that although the precipitating insult might vary anatomically from the level of the neck of the humerus to that of a Colles’s fracture the ischaemia was always confined to the forearm. He described the pathological abnormality as an infarct of the muscles and nerves of the forearm, which varied in size, in shape and in severity but was remarkably constant in its site; this applied to contractures in the leg as well as to those in the arm.

The muscles in these sites are confined within osteofascial compartments: the anterior tibial, the peroneal, and the deep posterior compartments of the leg, and the flexor and the extensor compartments of the forearm. It can be shown that this is of fundamental importance.

**Experiment 1.** In four warm human cadavers normal saline was introduced in increments of 25 millilitres into the anterior tibial compartment, the front of the thigh, the flexor aspect of the forearm and the front of the upper arm, and the pressure achieved was measured with a manometer by the technique previously described (Holden 1974). The results (Figs. 2 and 3) showed that in the distal

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**Fig. 2**

Comparison of rise in pressure after injection of saline into cadaveric muscle.

**Fig. 3**

Comparison of rise in pressure after injection of saline into cadaveric muscle.
segment of a limb it was possible to achieve a rise in the hydrostatic pressure with alarming ease but that this did not occur in the more proximal segment of the same limb.

Insults that cause an ischaemic contracture appear to be of two main types. Type I is the classical case of a major arterial injury occurring above the knee or above the elbow with the ischaemia centred in the distal segment. Type II is a direct insult to the distal segment, but the ischaemia still develops in that distal segment. Examination of the infarct produced in both types of contracture shows a constant pathology regardless of the site or of the nature of the precipitating insult. All these insults have the ability to make soft tissues swell.

Harman (1948) showed experimentally that in rabbits a tourniquet-induced ischaemia made the muscles swell after the release of the tourniquet. He quantified this swelling by comparing the weights of ischaemic muscles with those from the normal limb. After two hours of tourniquet-induced ischaemia the muscles increased in weight by 35 per cent, and after three hours by 50 per cent: such swelling was completely reversible. After four hours of ischaemia, although the amount of swelling that was produced was no greater than after three hours, he found that the ischaemia was perpetuated, and if animals were killed fourteen days after such an insult they had developed a permanent contracture. Harman could not explain the mechanism of this perpetuation.

Normally, fluid exchange through the capillary wall is effected by the filtration force of the capillary blood pressure forcing fluid and crystalloids out into the tissue fluid. Counteracting this is the osmotic differential of the plasma proteins drawing fluid back into the capillary (Fig. 4). At the arterial end of the capillary the filtration force exceeds the osmotic force and so fluid leaves the capillary. At the venous end the position is reversed and fluid returns.

After tourniquet-induced ischaemia (Fig. 5) there is a reactive hyperaemia so that the filtration force increases. There is also anoxic damage to the capillary wall so that plasma colloids leak out and the osmotic differential decreases. Much more fluid leaves the capillaries than returns and there is an increase in the volume of the tissue fluids. Where the tissues are confined in compartments this increase in volume will cause a rise in pressure in the tissues. As the pressure rises the venous end of the capillary will become occluded and eventually the whole capillary, and even the arterioles, may become occluded. A stage will be reached where almost no fluid returns to the capillary. Harman (1948) described this state of affairs: “It appeared as if the liquid component of the blood had filtered off almost completely”. As the microcirculation in the soft tissues becomes occluded the ischaemia becomes increasingly severe and a vicious circle occurs (Fig. 6). Once this is established it cannot be broken by relieving the original insult; only a prompt and generous fasciotomy will allow the tissues to swell without a dangerous rise of pressure.

It can be shown that the anterior tibial compartment of the rabbit is covered with a dense deep fascia, and by instilling fluid into it a rise in pressure can be built up in exactly the same way as in the human cadaver.
Experiment 2. Harman's tourniquet experiments were repeated in a series of forty-eight adult New Zealand white rabbits. A rubber tourniquet being applied to the left thigh for periods of four hours or longer. In twenty-four rabbits the tourniquet was released at the end of the ischaemic period and the tissues allowed to swell for a period of three hours. Then 4% per cent brodhenol blue solution was injected intravenously in a dosage of 3 millilitres per kilogram body weight to stain those tissues with a functioning capillary circulation (Clark and Blomfield 1945). The animals were then killed by an overdose of Nembutal and the muscles of the anterior tibial compartment examined and compared with the anterior tibial muscles of the normal side. In all cases the ischaemic muscles showed evidence of considerable oedema and swelling. In three rabbits all the anterior tibial muscles appeared to be stained normally. In four rabbits there was a patchy staining of the muscle fibres, some parts of some fibres being stained darkly and some not being stained at all. In seventeen rabbits the central mass of the muscles failed to stain at all though the more peripheral parts stained quite well. In many a central oval ischaemic area was produced similar to that in Seddon's model.

In the other twenty-four rabbits a tourniquet was applied to the left thigh for four hours, and then a generous fasciectomy was performed on the anterior tibial compartment. The wound was then sutured and three hours allowed for swelling to occur. At the end of this time an identical intravenous injection of brodhenol blue was made. Examination of the anterior tibial muscles of the ischaemic side failed to demonstrate any ischaemic area in any of the anterior tibial muscles in any of the rabbits. It appeared that performing a fasciectomy at the time of releasing the tourniquet prevented the development of an infarct.

I think that the perpetuation of the ischaemia caused by the swelling of limb muscles and nerves after injury is due to these tissues being contained in an unyielding compartment, and that this accounts for the 'selective' ischaemia that is the basis of all ischaemic contractures. An ischaemic contracture can develop from a multitude of different insults provided that the insult causes swelling of the soft tissues which are contained in such compartments. Such a concept would explain the constancy of site and the constancy of the pathological findings in the muscle and the nerve damage seen in ischaemic contractures, and demands that diagnosis of such ischaemia be made at a time when fasciectomy could relieve the tension before the ischaemia has become perpetuated.

**DIAGNOSIS**

Ischaemia is a relatively uncommon complication of injury to a limb, but its effects can be so devastating that it should be sought in every case. Eternal suspicion is necessary if ischaemia is not to be missed in the early stages. It is essential to think anatomically, and to observe the distal segments of the limb in which the ischaemia may be occurring.

Pain is the overriding symptom. It is persistent, progressive and unrelied by immobilisation. It is aggravated by passive stretching of the ischaemic muscle. Rarely, it may be absent altogether, but this is usually in those cases where there has been extensive nerve damage.

Nerve is the tissue most sensitive to ischaemia. and the most important physical sign is an increasing neurological deficit in those nerves that pass through the affected compartment. All too often an opinion on the circulation in muscle has to be formed from examination of five digits that protrude from the end of a plaster. Is the sensation normal in these digits? Can they be actively moved? Their colour and temperature is much less helpful. as compartmental ischaemia may be severe but yet may in no way diminish the circulation in the skin.

The pulse may be difficult enough to elicit but even more difficult to interpret. Blount (1950) summarises this difficulty: 'The pulse is unreliable as a danger signal. Its absence is not an indication for operation nor its presence a guarantee that ischaemia will not develop.' The distal pulse is rarely obliterated by compartmental swelling yet the circulation in muscle and nerve is at a standstill. Similarly, a good peripheral pulse may easily be detected when the major limb vessel has actually been transected more proximally (Patman, Poulos and Shires 1964). Conversely, a distal pulse may be totally absent. but there is a considerable body of informed opinion which suggests that provided this is the only abnormal physical sign and that there is no pain on full passive dorsiflexion and normal sensation and muscle power distally, then there is no indication to explore the vessel. Such a patient will, of course, be repeatedly observed by the surgeon personally to detect any deterioration.

Whenever possible. the soft tissues should be palpated. Are they tender? Are they swollen? Are they tense? Where the diagnosis of an arterial injury is made the artery itself must be explored and repaired, and the distal compartments decompressed if there is any doubt about their tension. In the Type II cases prompt and generous fasciectomy alone should relieve the ischaemia.

The diagnosis of both types of ischaemia in an injured limb can usually be made on clinical examination alone. Investigations such as arteriography are time-wasting and unnecessary in most cases. It is possible to measure intracompartamental pressure using the technique of Whitesides et al. (1975), but the decision to perform a fasciectomy should usually be a clinical one. Clinical judgement must be based on a high degree of suspicion. eternal vigilance and a clear understanding of the pathological abnormalities that may be present.

It should now be possible to avoid ischaemic contractures in all but the most severely mutilating injuries.

**REFERENCES**
